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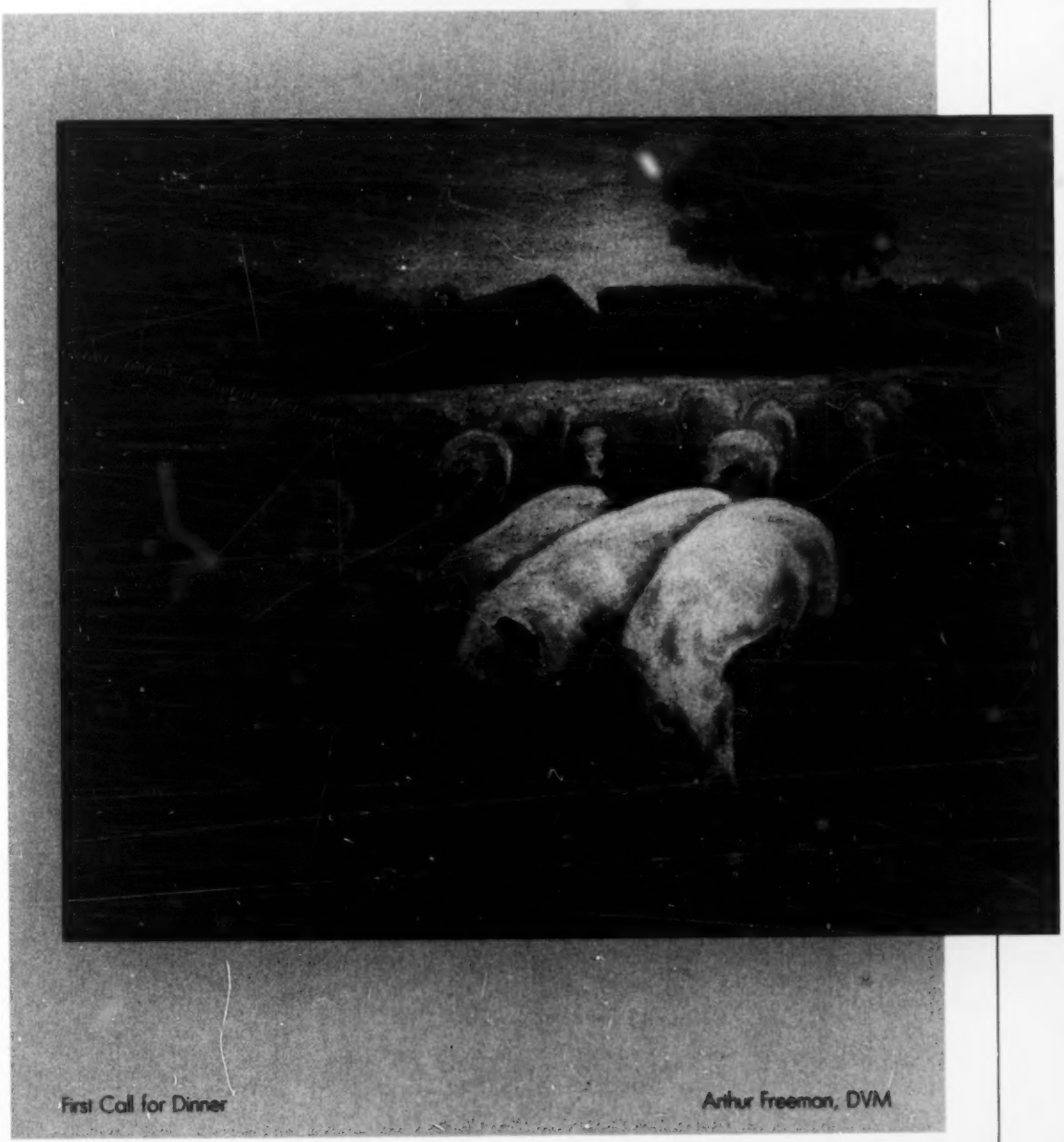

First Call for Dinner — Arthur Freeman, DVM

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About the cover

Dr. Arthur Freeman was born in Youngstown, Ohio, and received his DVM at Ohio State University in 1955. Four years later, he accepted a position as assistant editor at the American Veterinary Medical Association where, in 1972, he was appointed editor-in-chief; it was originally Dr. Freeman's idea to use artwork featuring animals on the covers of the *JAVMA*. From 1985 to his retirement in 1989, he served as executive vice president of the AVMA. Dr. Freeman now resides in Carmel, Ind.

People associated with the veterinary profession, via their work or family relation to a veterinarian, who wish to have their animal art considered for reproduction on the cover of the *JAVMA* should send a color photograph (no larger than 5″ × 7″) to Dr. Janis Audin, AVMA, 1931 N Meacham Rd, Suite 100, Schaumburg, IL 60173-4360.

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BPA

Prevalence of antibody to caprine arthritis–encephalitis virus in goats in the United States

Randall C. Cutlip, DVM, PhD; Howard D. Lehmkuhl, PhD; Jerome M. Sacks, PhD; Amy L. Weaver, MS

Summary: *Goats from 28 states were tested for antibodies to caprine arthritis-encephalitis virus. Of 3,790 goats, 1,175 (31%) tested positive, and of 196 herds tested, 143 (73%) had 1 or more seropositive members. This prevalence, based on serum samples from all goats in the participating herds, was lower than most rates reported in other studies. Such studies were based on fewer samples, incomplete sampling of herds, or smaller geographic base. Prevalence was highest in western Pacific and northern plains regions, increased with age to 3 years, was highest among goats on family-owned farms, and was lowest in the Angora breed. Differences in prevalence were not related to gender or size of herd.*

Caprine arthritis-encephalitis (CAE) is a multisystemic disease of goats, caused by a lentivirus. The disease is most commonly recognized as chronic lymphocytic and degenerative polyarthritis, interstitial mastitis, or chronic encephalomyelitis of adult goats, but occasionally is seen as acute lymphocytic leukoencephalomyelitis of neonatal goats.[1-5] Many infected goats do not manifest clinical signs of disease, but are persistently infected and are positive for specific antibody.

Two serologic studies of the prevalence of CAE viral infection in goats in the United States have been reported.[4,6] A study of 1,160 goats from 24 states indicated prevalence of 81%.[4] However, results were considered biased because 836 of the 1,160 samples were submitted for diagnostic purposes from herds with clinical evidence of arthritis.[7] The other 324 samples were from 19 herds in Washington and Idaho. A study of goats from 13 dairies in California indicated prevalence of 53% in 2,826 goats, with range of 39 to 80% among herds.[6] Some of these goats were involved in a program for eradication of the CAE virus.

From the National Animal Disease Center, Agricultural Research Service, United States Department of Agriculture, PO Box 70, Ames, IA 50010 (Cutlip, Lehmkuhl, Sacks) and the Department of Statistics, Iowa State University, Ames, IA 50011 (Weaver).

The authors thank Dr. Joan Bowen and Keith Sherman for technical assistance.

The objective of the study reported here was to test 3,790 goats from 28 states for antibodies to CAE virus. Our objective was to expand over previous studies the number and distribution of goats tested to obtain a more comprehensive estimate of the prevalence of CAE virus infection in the United States.

Materials and Methods

Blood samples were collected from 196 herds of goats in 28 states. From 1982 to 1988, samples were collected by the owners or local veterinarians from 3,790 goats aged 4 months through 15 years. In most instances, the entire herd was tested. Herds were selected because of the interest of owners in a pilot field eradication study. At the time of blood sample collection, the goats were not involved in an organized control program although they were being selectively culled by the owner. Comparisons were made among regions, states, age, gender, breed, and owner type.

States were grouped into 6 regions: northern Atlantic, comprising Connecticut, Maine, New Jersey, New York, Vermont, and West Virginia; northern plains, comprising Iowa, Illinois, Indiana, Kansas, Michigan, Minnesota, Missouri, Nebraska, Ohio, and Wisconsin; Rocky Mountain, comprising Colorado, Montana, and Wyoming; southern Atlantic, comprising Arkansas, Georgia, Louisiana, North Carolina, and Tennessee; southern plains, with the single state of Arizona; and western Pacific, including Oregon and Washington. Only states with at least 5 goat herds were compared individually.

Serum samples were tested for precipitating antibodies to CAE virus, using an agar gel immunodiffusion test described for detection of ovine progressive pneumonia virus.[8] Minor alterations in the procedure consisted of using 7 ml of 0.8% agarose containing 0.4% polyethylene glycol (MW 6,000) rather than 6 ml of 1% agarose without polyethylene glycol as originally reported. Antigen was the ovine progressive pneumonia virus isolate WLC-1 prepared as described.[8] Antigenic cross-reactivity has been documented between the glycoprotein and the internal proteins of ovine progressive pneumonia and CAE viruses[9] (unpublished

Table 1—Prevalence of goats with antibody to caprine arthritis-encephalitis (CAE) virus among regions, states, ownertypes, and herd size

Group	Proportion of goats seropositive (%)	No. of goats tested	Proportion* of herds seropositive (%)	No. of herds tested	Mean† group size
Region‡					
Northern Atlantic	24	654	78	18	36
Northern plains	41	1,210	68	81	15
Rocky Mountains	25	1,077	78	64	17
Southern Atlantic	22	659	63	16	41
Western Pacific	49	87	00	10	9
State§					
Arkansas	44	216	80	5	43
Arizona	49	103	71	7	15
Colorado	28	703	76	55	13
Iowa	45	261	78	18	15
Illinois	48	62	100	6	10
Kansas	36	140	60	5	28
Michigan	4	48	17	6	8
Minnesota	49	254	88	8	32
Missouri	44	70	100	5	14
Montana	21	322	88	8	40
North Carolina	54	24	50	6	4
Nebraska	21	152	63	8	19
New York	34	236	71	7	34
Ohio	29	98	67	12	8
Oregon	68	19	100	5	4
Washington	44	68	100	5	14
Wisconsin	74	102	50	10	10
Other‖	15	912	70	20	46
Owner type					
Farm family	36	2,812	73	190	15
Private institution	12	472	75	4	118
Public institution	19	513	100	2	257
Herd size					
1 to 10	33	488	57	115	4
11 to 50	35	1,344	95	66	20
> 51	27	1,958	100	15	131
All	31	3,790	73	196	19

*Herds with at least 1 seropositive goat. †Mean number of goats in each category within different groups. ‡Southern Plains Region omitted because only Arizona was represented. §States with 5 or more herds tested. ‖States with less than 5 herds tested: Connecticut, Georgia, Indiana, Louisiana, Maine, New Jersey, Pennsylvania, Tennessee, Vermont, West Virginia and Wyoming.

observations). Test results were read as positive only when complete lines of identity were formed with a known positive serum. Only 13% of the sera produced incomplete lines and were considered negative for the purpose of this study. When some such goats were tested 1 or more years later, 49% had positive results and 22% continued to have suspicious results. Results were analyzed as percentage of seropositive goats and as percentage of herds with seropositive members. Prevalence rates for breeds and their herdmates were computed for herds with more than 1 breed.[10,11] Herdmate comparisons were also used for other categories as needed.

Results

Prevalence of goats with antibody to CAE virus varied among regions and among states, but association with herd size was not apparent (Table 1). Among individual herds, seropositivity ranged from 0 to 100%. Size of herds with all seropositive goats ranged from 1 to 56, and that of herds with no seropositive goats ranged from 1 to 17. Prevalence of goats with specific antibody among family-owned farm herds was about twice the rate of public or private institutional herds (Table 1). This difference was observed in all age groups from 0 to 6 years. Mean ± SD difference was 21 ± 6% (*t* test; $P < 0.01$).

Age, gender, or breed identification was missing for approximately 30% of the goat records, but prevalence of goats in the unknown age, gender, or breed categories (Table 2) did not differ greatly from the overall mean of 31%. Prevalence of goats with antibody to CAE virus increased with age (Table 2). Mean increase, approximately 14%/yr, based on a linear regression analysis, was statistically significant ($R^2 = 74\%$; $P < 0.01$). Average herd size decreased by approximately 22%/yr, based on linear regression analysis ($R^2 = 96\%$; $P < 0.01$). Females and sexually intact males had almost identical average prevalence and age specific rate patterns. In wethers, prevalence was less than half that in sexually intact males, but compared with herdmates, the difference was only 11 ± 8% and was not statistically significant. Prevalence among breeds of goats with specific antibody was similar, except for the 9 herds of Angora that had approximately a third the average prevalence. Computation of herdmate differences confirmed low prevalence in the Angora breed, (*t* test; $P < 0.01$; Table 3).

Prevalence of goats with antibody to CAE virus was computed for each herd, and herds were clas-

Table 2—Prevalence of goats with antibody to CAE virus among age groups, genders, and breeds

Group	Proportion of goats seropositive (%)	No. of goats tested	Proportion* of herds seropositive (%)	No. of herds tested	Mean† group size
Age (years)					
<1	18	610	47	75	8
1	28	838	63	116	7
2	34	525	74	91	6
3	45	281	66	77	4
4	41	217	75	60	4
5	48	115	67	39	3
≥6	43	182	65	84	2
Gender					
Female	33	2,463	81	146	17
Male	34	232	58	77	3
Wether	14	191	33	24	8
Unknown	27	904	51	69	13
Breed					
Alpine	39	473	69	42	11
Angora	11	28	22	9	3
LaMancha	39	87	67	9	10
Nubian	32	823	72	81	10
Oberhasli	25	172	100	3	57
Pygmy	37	19	50	6	3
Saanen	37	378	67	30	13
Toggenburg	33	322	72	29	11
Crosses	22	322	55	49	7
Unknown	27	1,166	60	91	13

*Herds with at least 1 seropositive goat. †Mean number of goats in each category.

Table 3—Prevalence of antibody to CAE virus among goat breeds and among members of herds comprising more than 1 breed

Breed	No. of herds tested	Proportion of breed seropositive (%)	Proportion of herdmember seropositive (%)	Herdmember difference (SEM; %)
Alpine	34	39	37	02 (04)
Angora	9	7	37	−30* (07)
LaMancha	8	25	29	−04 (09)
Nubian	52	31	24	07 (04)
Oberhasli	2	57	71	−14 (72)
Pygmy	4	23	23	0 (18)
Saanen	25	28	36	−08 (06)
Toggenburg	25	46	35	11 (07)
Crosses	44	28	31	−03 (05)

*Herdmember difference is significant ($P < 0.05$).

Table 4—Distribution of female and male goats in herds of low, medium, and high seroprevalence to CAE virus

Relative prevalence	No. of herds	Proportion seropositive (%)	Average herd size
Females			
Low*	85	6	20
Medium†	31	32	15
High‡	78	69	15
Male			
Low*	36	2	3
Medium†	18	42	4
High‡	23	87	3

*Herd prevalence > 0.5 SD below group mean. †Herd prevalence within 0.5 SD of group mean. ‡Herd prevalence > 0.5 SD above group mean.

sified as low, medium, or high prevalence if values were at least 0.5 SD below the mean, within 0.5 SD of the mean, or at least 0.5 SD above the mean, respectively (Table 4). This procedure, which should classify the herds into 3 approximately equal groups (31, 38, and 31%) on the basis of a normal distribution, revealed obvious clustering. For either gender, most of the herds were the high or low prevalence groups. The medium prevalence group had the fewest numbers of herds.

Discussion

In this serologic study of the prevalence of antibody to CAE virus in goats in the United States, we have documented the overall prevalence to be about half that previously reported.[4,6,7] This difference probably relates to our testing of younger goats, all goats in most herds, more clinically normal goats, and a wider distribution of goats. Our finding of 49% prevalence in the western Pacific region closely parallels the 53% prevalence reported in goats in California.[6] Prevalence of seropositive goats among states was within a factor of 2 except for Indiana and Michigan, which had the lowest prevalence, and for Oregon and Wisconsin, which had the highest prevalence. The high prev-

alence rate (74%) in Wisconsin was attributable mostly to 1 herd of 56 goats in which 100% of the members were seropositive. The remaining 9 herds of 46 goats had 41% seropositivity. Prevalence in the western and middle parts of the country was about twice that in the eastern and Rocky Mountain areas of the country. As reported in previous studies, prevalence increased with age; however, in our study, leveling-off was evident after 3 years of age. The reason prevalence increased with age is not known, but may have resulted from lateral transmission of virus between adults or from delayed seroconversion after virus was transmitted to neonates. The plateauing of seroprevalence at 3 years of age may be the result of the owner culling clinically affected goats. The 2 to 3 times higher prevalence in family-owned farm herds than in other owner types may be related to greater movement of goats among family farm owners, as well as other management practices. Differences in prevalence among breeds was not significant except for the lower prevalence in the Angora breed. Lower prevalence in the Angora breed was reported from Australia.[12] To our knowledge, prevalence in Pygmy goats has not previously been reported, but in this study, paralleled that seen in most other breeds.

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Book Review: A Concise Survey of Animal Behavior

This book is a short overview of animal behavior, with a focus on acquainting the reader with the basic fundamentals of ethology. The authors use the first chapter to review the history of the study of animal behavior from Greek philosophy to the work of early 20th century scientists. The second chapter outlines the concepts used by ethologists: behavior as adaptation to an environment, evolution as it may explain the appearance of a particular behavior, mechanisms controlling the initiation of behavior, and how a behavior develops and changes. The next chapter addresses some common ethologic research areas. These include: biological rhythms, migration, hormonal control of behavior, and social behavior. Brief discussions are also included regarding the issues of animal reasoning, animal rights, and human–animal interactions. The final chapter gives the reader an ethologist's view of the most important aspects of normal behavior for domestic ruminants, nonruminants, carnivores, birds, and exotic pets. A supplemental reading list is provided at the end of the book referencing a number of *Scientific American* articles of historical interest.—[*A Concise Survey of Animal Behavior. By E.K. Honore and P.H. Klopfer. 186 pages; illustrated. Academic Press Inc, 1250 Sixth Ave, San Diego, CA 92101. 1991. Price $29.95*]—WALTER F. BURGHARDT, JR.

Prevalence, type, and importance of splenic diseases in dogs: 1,480 cases (1985–1989)

W. L. Spangler, DVM, PhD, and M. R. Culbertson, DVM, PhD

Summary: *The prevalence data of splenic diseases from 3 sources were studied. Group 1 consisted of a general diagnostic survey of accessions submitted from private veterinary hospitals in California during a period of approximately 4 years and included 1,372 submissions of canine splenic tissue. Group 2 consisted of surgical splenectomy specimens from 92 dogs; the specimens were submitted to the laboratory for gross and histologic evaluation prior to fixation, and a questionnaire was subsequently sent to determine the outcome of the disease. Group 3 was made up of specimens of 105 splenic lesions derived from a large colony of Beagles with complete medical records and records of pathologic findings.*

In this study, splenic hematoma and hyperplastic nodule, not hemangiosarcoma, made up the bulk of splenic lesions. Hemangiosarcoma was the most frequent neoplasm of the canine spleen, but the combined prevalence of all other splenic neoplasms was similar to that of hemangiosarcoma alone. Splenic hematoma and hemangiosarcoma were grossly indistinguishable in most cases. Hyperplastic lymphoid nodules and hematomas of the spleen appeared to represent a continuum. If that finding was correlated with microscopic splenic blood flow, specific causal relationship could be suggested. Prognostically, the live/dead ratio and mean survival of dogs with various splenic lesions differed significantly.

Most clinical reports that specifically consider the canine spleen are concerned with some aspect of neoplasia, and often more specifically, with hemangiosarcoma.[1-5] In a report including hemangiosarcomas in all tissues, the prevalence of canine hemangiosarcoma varied between 0.3 and 1.98%. Among hemangiosarcomas reported, 62% originated in the spleen. This perspective of splenic disease in dogs tends to distort the importance of neoplasia in the canine spleen relative to the prevalence and importance of nonneoplastic splenic diseases.[3,5] Although actual prevalence data concerning specific primary splenic diseases are scant, some publications emphasize hyperplastic (nodular lymphoid hyperplasia) and degenerative (splenic hematoma) lesions.[3,5,6] The purpose of the study reported here was to determine the prevalence of specific splenic diseases in dogs, as well as possible pathogenetic mechanisms for the formation of splenic hematoma.

From the California Veterinary Diagnostics Inc, 3911 W Capitol Ave, West Sacramento, CA 95691.

Supported in part by the Office of Health and Environmental Research, US Department of Energy center contract DE-AC03-76SF00472 and under grant DE-FG03-89ER60914/92 with the University of California, Davis.

Criteria for Selection of Cases

The dogs in this study were allotted to 3 groups. Records of group-1 dogs were derived from retrospective review of computerized pathologic records from a private diagnostic laboratory.[a] The records were searched for diagnoses that included spleen or splenic as part of the diagnostic terminology. A total of 1,372 cases in dogs were retrieved from a period of 50 months (July 1985 to September 1989). These cases represented 1.3% of approximately 124,000 total accessions from all species of animals during that period. The selected cases represented canine surgical/necropsy specimens submitted from veterinary practices distributed throughout California. Representative sections from all fixed materials were selected from individual submissions and prepared for microscopic evaluation. Group-2 dogs consisted of 92 dogs, for which unfixed spleens derived from surgical splenectomy, were submitted to the laboratory for gross and histologic evaluation (follow-up subgroup). Although the specimens from those dogs were part of the spleens considered in group 1, these tissues were examined grossly by one of the authors (WLS), described, measured, and sampled at multiple (minimum of 7) sites from 0.5- to 1-cm serial gross sections obtained from the fresh tissue. Photographs were obtained in many cases. Microscopic evaluation of these specimens was followed by a letter to the submitting veterinarian requesting the specific health status of the animal and any omitted information (eg, age, gender, breed) to

[a]California Veterinary Diagnostics Inc, West Sacramento, Calif.

Table 1—Prevalence of canine splenic lesions by specific diagnosis (group 1)*

Lesion	No.	Percentage
Hyperplastic nodule	306	23
Hemangiosarcoma	137	10
Hematoma	129	10
Hyperplastic nodule/ hematoma	128	10
Accessory spleen	46	3
Siderotic plaque	38	3
Lymphosarcoma	38	3
Diffuse lymphoid hyperplasia	22	2
Leiomyosarcoma	23	2
Undifferentiated sarcoma	23	2
Mixed lymphoid myeloid hyperplasia	17	1
Myeloproliferative disease	17	1
Myeloid metaplasia	15	1
Splenic infarction	14	1
Splenic arterial thrombosis	12	<1
Metastatic carcinoma	11	<1
Myelolipoma	8	<1
Splenic torsion	8	<1
Fibrosarcoma	7	<1
Mast cell tumor	6	<1
Splenitis	6	<1
Metastatic adenocarcinoma	5	<1
Myeloid hyperplasia	5	<1
Plasma cell myeloma	4	<1
Splenic abscess	4	<1
Reticuloendothelial hyperplasia	4	<1
Amyloidosis	3	<1
Multidifferentiated sarcoma (mesenchymoma)	3	<1
Myxosarcoma	2	<1
Hemangioma	2	<1
Osteosarcoma	1	<1
Sinusoidal telangiectasis	2	<1
Necrosis/hemorrhage	2	<1
Splenic fibrosis	2	<1
Capsulitis	2	<1
Capsular mineralization	1	<1
Traumatic rupture	1	<1
Histiocytosis	1	<1
Extramedullary hematopoiesis†	84	6
Congestion†	97	7
Lymphoid atrophy/depletion†	49	4
Hemosiderosis†	20	2
Lymphoid necrosis†	17	1
Splenic necrosis†	4	<1

*Forty-six submissions characterized histologically as normal spleen are excluded from this table. †Represents the diagnosis in the spleen derived from cases in which multiple tissues were submitted for evaluation.

*Table 2—Prevalence of canine splenic lesions by specific diagnosis (group 2)**

Lesion	No.	Percentage
Hemangiosarcoma	22	24
Hyperplastic nodule/hematoma	18	19
Hematoma	18	19
Hyperplastic nodule	6	6
Myeloid metaplasia	5	5
Leiomyosarcoma	4	4
Multidifferentiated sarcoma (mesenchymoma)	4	4
Histiocytic sarcoma	3	3
Lymphosarcoma	3	3
Hemangioma	2	2
Splenic infarction	2	2
Congestion	2	2
Splenic vascular thrombosis	1	1
Fibrosarcoma	1	1
Plasma cell myeloma	1	1
Myxosarcoma	1	1

*Ninety-two individual submissions, 93 diagnoses.

Table 3—Prevalence of splenic disease in Beagles (group 3)*

Lesion	Total	Percentage
Hyperplastic nodule	52	48
Diffuse lymphoreticular hyperplasia	12	11
Hemangiosarcoma	10	9
Lymphosarcoma	7	6
Hyperplastic nodule/hematoma	6	6
Metastatic neoplasia	6	6
Undifferentiated sarcoma	4	4
Myeloid metaplasia	5	4
Lipoma	2	2
Leiomyosarcoma	2	2
Hemangioma	2	2

*Three hundred seventy-four dogs at risk.

statistically evaluate clinical prognostic factors in canine splenic disease. In group-3 dogs, the surgical and necropsy records from a colony of Beagles were reviewed for similar purposes. These dogs were subject animals in a life-time study of chronic exposure to ^{90}strontium or ^{226}radium. In that study,[7] animals were exposed to various doses of radionuclides with various routes of administration and were monitored for life by periodic clinical and radiographic examination as well as by a variety of clinical analytical methods as required by the protocol. Complete necropsy was performed on all (865) dogs. The data concerning splenic diseases in 105 of the dogs were derived from retrospective analysis of the slides and records of pathologic findings, which for the most part were originally generated by the authors.

Information derived from these 3 data sources was tabulated separately. Each of the splenic lesions (diagnoses) was reviewed histologically. The resulting information was then manually entered into a flat data computer file capable of segregating, grouping, and ordering the data for subsequent analysis. The duration of postoperative survival in the group-2 dogs was compared statistically by use of the Mann-Whitney method. Gender proportions were tested by use of the χ^2 method.

Results

Hyperplastic nodules and splenic hematoma were the most often encountered disease conditions among the canine spleens from all 3 data sources considered by this study. Splenic hemangiosarcoma was second in overall prevalence. In our data, the ratio of nonneoplastic to neoplastic disease in the canine spleen (the general diagnostic population) was 3.6:1 (1,039 to 287) in group 1 (Table 1), 1.3:1 (52 to 41) in group 2 (the follow-up subgroup; Table 2), and 2.3:1 (75 to 33) in group 3 (Beagles; Table 3). Splenomegaly resulted from nonneoplastic diseases in 56% of diagnoses (52 of 93) and from splenic neoplastic diseases in 44% (41 of 93) of diagnoses in group 2. Also in group 2, splenomegaly resulting from hyperplastic nodule and hematoma formation accounted for 42 cases (46% of splenic submissions), whereas splenomegaly and splenectomy attribut-

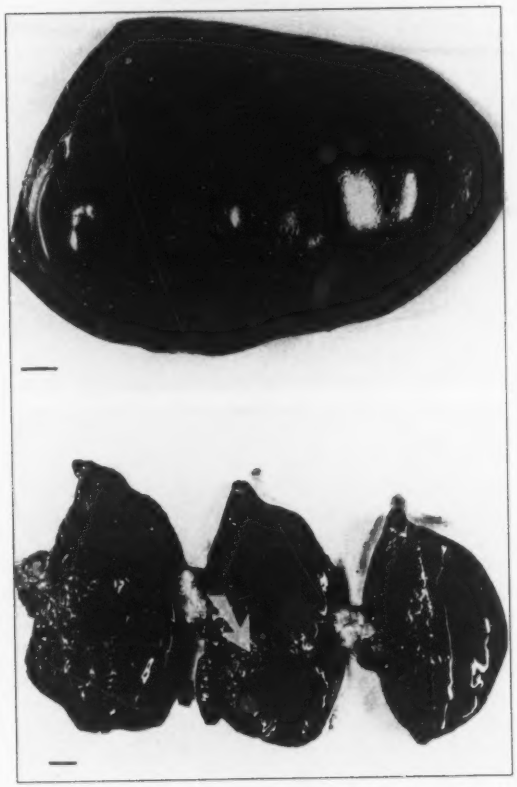

Figure 1—A 10.5-cm subcapsular splenic hematoma (top). Cross sections of a subcapsular hematoma demonstrating a histologically confirmed underlying focus of nodular lymphoid hyperplasia (arrow; bottom). Bar = 1 cm.

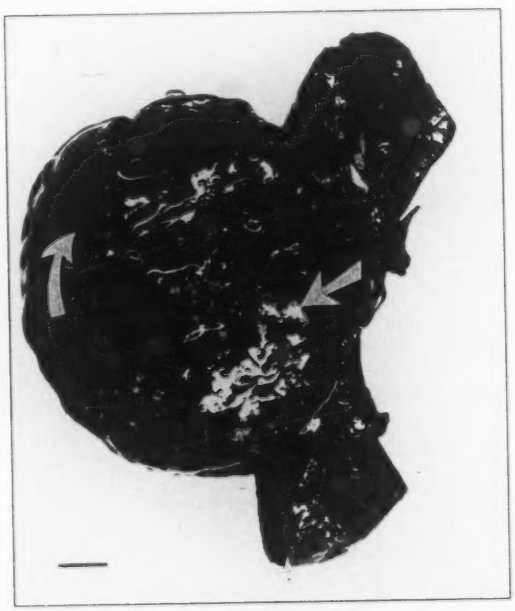

Figure 2—Sectioned surface of a splenic nodule with a discrete area of nodular lymphoid hyperplasia (histologically confirmed; straight arrow) underlying an organizing hematoma (curved arrow); bar = 1 cm.

able to hemangiosarcoma accounted for 22 cases (24% of splenic submissions).

Hyperplastic lymphoid nodules were observed in many specimens in association with splenic hematoma. Macroscopic and microscopic examinations revealed disorganized or compressed lymphoid hyperplastic, or mixed lymphoid-hematopoietic foci along the margins of a splenic nodule consisting of hemorrhage, organizing fibrin, or degenerating erythrocytes (Fig 1 and 2). Similarly, hemorrhagic areas were found within some nodular lymphoid or hematopoietic foci (Fig 3). In some specimens, only erythrocytes or the sequelae of prior hemorrhage (organizing clot) could be detected microscopically (splenic hematoma).

There was no typical gross appearance to reliably distinguish splenic hematoma from hemangiosarcoma and most generally, pathologic features were similar. However, primary hemangiosarcoma of the spleen tended to be multifocal, nodules were individually smaller than hematomas, and most were cavitated with a honeycomb appearance on the sectioned surface. Many of the cavities in hemangiosarcoma contained unclotted blood and resulted in spherical nodules of various sizes, many of which were dimpled or collapsed with a slack or wrinkled overlying splenic capsule, indicating a lack of underlying structural integrity. In contrast, the clotting of blood and subsequent organization tended to give splenic hematomas a firm consistency, producing a nodule that smoothly and firmly distorted the surface of the spleen and one, which on the cut surface, appeared to be compartmentalized or geometrically divided into compartments of various sizes with straight sides or walls made up of connective tissue and with brown discolored irregular areas of necrosis and tissue liquefaction.

The German Shepherd Dog breed ranked first in breed prevalence among the splenic diseases in the categories of hyperplastic nodule/hematoma, hemangiosarcoma, and lymphosarcoma. Golden Retrievers and Labrador Retrievers ranked second and third, respectively, in those categories. An attempt was not made to correct these data for actual breed prevalence in this area. Hyperplastic nodule/hematoma and hemangiosarcoma were found at virtually the same mean age in dogs (10.5 and 10.4 years, respectively) and were evenly distributed between the genders. In group 2, the mean age of dogs with splenic lymphosarcoma was 9.4 years, but the diagnosis of accessory spleen was made at 6.8 years. Myeloid metaplasia/hyperplasia was found in dogs with a mean age of 8.7 years and was found 5 times more often in females than males ($\chi^2 = 6.24$; $P < 0.025$).

Foci of accessory or ectopic splenic tissue represented 3% of splenic lesions submitted to the laboratory (Table 1). These aggregates of viable and identifiable splenic tissue were of various sizes, remote from the spleen, and most were implanted in or attached to the omentum.

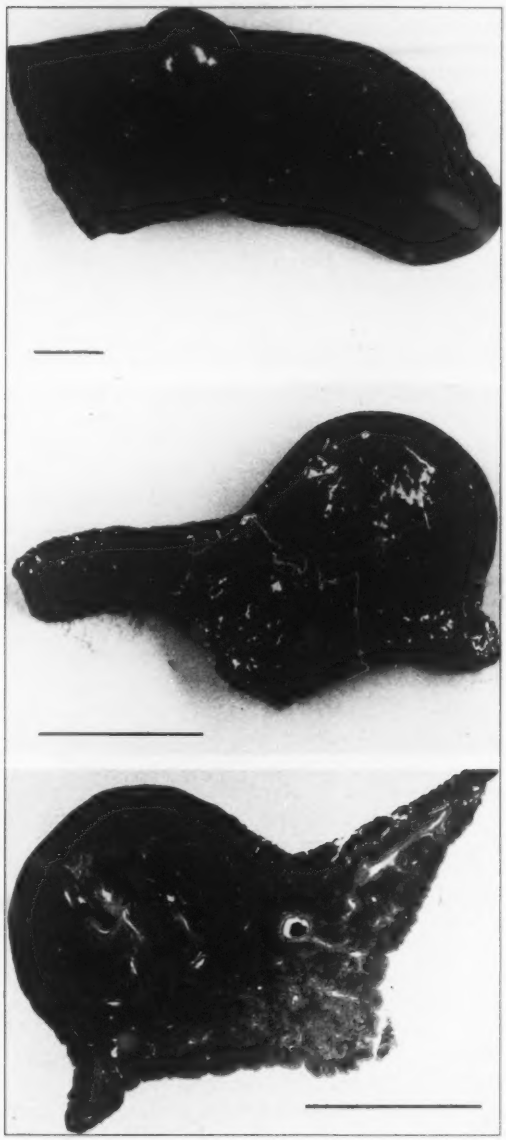

Figure 3—A 1.5-cm single black nodular mass protruding from the parietal surface of the spleen (top). Sectioned surface of splenic nodule in top photograph. The sectioned surface is homogeneous with no distinguishing features (middle). Photomacrograph of section of spleen of a dog. Notice nodular hyperplasia of splenic lymphoid elements with widely dispersed hemorrhages (arrows; bottom). H&E stain; bar = 1 cm.

Myeloid metaplasia/hyperplasia was found as a cause of diffuse, nonnodular splenomegaly in dogs and was characterized by diffuse proliferation of hematopoietic elements at the expense of normal lymphoid follicular and sinusoidal architecture. This proliferation was diffuse throughout the splenic parenchyma, with no organization as discrete hematopoietic foci such as that observed in cases of splenic extramedullary hematopoiesis responding to severe anemia.

Neoplastic disorders of the canine spleen were dominated by hemangiosarcoma in all groups. Hemangiosarcoma accounted for 48% (137 of 287) of all splenic neoplasia in group 1, whereas lymphosarcoma constituted 13% (38 of 287) of the diagnoses in the neoplastic category (Table 1). The remaining 39% (112 of 287) of neoplasms was divided among 13 distinct histologic types.

Definitive survival information was obtained for all but one of the 92 cases in the follow-up survey (group 2). When live/dead ratios were calculated with mean survival times for all neoplastic splenic diseases, 68% of the dogs (28 of 41) were dead at the time of follow-up, and the mean survival was 3.13 months (n = 39). For all nonneoplastic splenic diseases, only 41% (21 of 51) of the dogs were dead at the time of follow-up, and the mean survival was 7.06 months (n = 51). More specific analysis revealed that 82% of dogs with a diagnosis of splenic hemangiosarcoma (18 of 22) were dead at the time of the follow-up (mean survival = 3.02 months), whereas only 37% of dogs with a diagnosis of hyperplastic nodule, hematoma, or both (16 of 41) were dead (mean survival = 7.55 months; $P < 0.001$). These data included all those dogs that died in the immediate postoperative period (0 months survival time). Results for nonangiogenic and nonlymphomatous sarcomas of the spleen were divergent. Although dogs with a diagnosis of splenic fibrosarcoma (1 of 92), leiomyosarcoma (4 of 92), histiocytic sarcoma (3 of 92), and splenic osteosarcoma (1 of 92) were all dead at the time of follow-up (mean survival = 2 months), those dogs with multidifferentiated sarcoma/mesenchymoma (5 of 92) were alive 7.33 months after surgery.

Discussion

Because the spleen is accessible through palpation and radiographic, cytologic, and surgical procedures and because of the high prevalence of splenomegaly detected in this study, we would rank examination of the spleen as an important overall consideration in the practice of veterinary medicine in dogs.

The prevailing perception among veterinarians is that hemangiosarcoma is the most prevalent form of canine splenic disease.[1-4] However, on the basis of more recent surveys,[5,6] and our findings, this perception of splenic disease may require reevaluation. It is only recently that the term hematoma has been used in reference to discrete splenic lesions.[5,6] On the other hand, nodular hyperplasia of lymphoid components is a recognized and frequently encountered lesion in the spleen of aging dogs.[8-10] Although the recognition that hemorrhage may occur in these areas of splenic nodular hyperplasia can be cited, it is anecdotal and not substantiated by numeric or anatomic data.[9,10] The complete pathologic evaluation of large numbers of

spleens (group 2) in this report, in which nodular hyperplastic lymphoid elements were observed with superimposed hematomas, represents convincing anatomic evidence that splenic hyperplastic nodules and splenic hematomas in dogs are related. This association is more strongly supported when the circulatory pattern through the canine spleen is considered.[11,12] A portion of the arterial blood arriving in the spleen through the branches of the follicular arteries enters the reticular spaces of the marginal zone surrounding splenic lymphoid follicules. This blood exits the marginal zone by draining via open-ended venous structures into the sinusoids of the red pulp and finally into collecting veins. It is easy to visualize the consequences of marginal zone distortion resulting from nodular lymphoid hyperplasia with regional disruption of splenic blood flow, failure of marginal zone circulation, and accumulation of blood within and around the hyperplastic nodule eventually leading to hematoma formation, hypoxia, and necrosis.

Although splenic hemangiosarcoma (n = 137) was the second most frequent diagnosis, the prevalence of splenomegaly associated with hyperplastic nodule/hematoma (n = 563) was approximately 4 times greater than hemangiosarcoma in the group-1 dogs (Table 1). In the more limited follow-up series of spleens (group 2), hyperplastic nodule/hematoma and hemangiosarcoma accounted for 45% (42 of 93) and 24% (22 of 93) of diagnoses, respectively; a ratio of 2:1 (Table 2). These observations were primarily responsible for initiation of the complete gross and histologic evaluation of splenic submissions in the group-2 dogs of this study and confirm the relationships between nodular enlargement of the spleen and the prevalence of nonneoplastic nodular splenomegaly. The prevalence of type and class of splenic disease varied in this study on the basis of the sampling method. In the case of the larger general diagnostic survey (group 1), all splenic submissions were combined without regard for the basis of the submission. Included in this segment of the population was a substantial number of multiple tissue submissions derived from necropsy on dogs of diverse ages and dying from a variety of infections or unknown specific causes. Also included in the general diagnostic survey were sections of spleens derived from abnormalities observed during exploratory laparotomy. Many of these were lesions lacking primary disease importance, and therefore, dilute the relative prevalence of primary splenomegaly. On the other hand, the follow-up survey (group 2) consisting entirely of splenectomy specimens is, in some ways, a more accurate representation of the prevalance of specific types of splenic diseases resulting in splenectomy in the general canine population. This conclusion is strengthened to some degree because one pathologist was responsible for gross evaluation and tissue selection from all specimens submitted for evaluation. The Beagle population (group 3) represents data from a tightly controlled population of purebred dogs. These data are most like that observed in group-1 dogs. It should be mentioned, however, that Beagles as a specific breed contributed in a minor way to the overall prevalence of splenic disease and total spleen submissions to the laboratory. This may be the result of a small number of Beagles found in the pet population of this geographic area.

Accessory splenic tissue made up 3% of submissions in the group-1 dogs (46 of 1,326). Although accessory spleen was noticed among group-2 dogs, it was not tabulated because the splenectomy specimen was generally accompanied by a variable amount of omentum, much of which was tightly adhered to the surface of nodular formations in the splenic parenchyma. The reason for the presence of accessory splenic tissue in most cases was obscure. Trauma is a reported cause,[8,13] and on the basis of our observations, fragmentation of splenic nodular lesions may be a major contributing factor. In our specimens, we were consistently unable to grossly distinguish between accessory splenic tissue and the omental implantation of hemangiosarcoma.

We could only find the descriptive pathologic features of myeloid metaplasia/hyperplasia as an unreferenced discussion in a standard pathology text.[14] It was an important cause of splenomegaly and splenectomy in all 3 groups in this study and by this inference, is apparently of clinical importance. A case report of a disease syndrome with striking similarities to these dogs was found.[15] Information in the follow-up survey revealed a lack of positive correlation between myeloid metaplasia and neoplastic myeloproliferative disease involving the spleen. In group-2 dogs, 5 cases of splenic myeloid metaplasia were recorded. Three of the 5 dogs were alive at the time of the survey with a mean survival at that point of 7.7 months (range, 4 to 13 months). One of the dogs was dead with a postmortem diagnosis of myeloproliferative disease, and one of the dogs was lost to follow-up. The gender of the animal was reported for only 13 of the 20 cases of myeloid metaplasia/hyperplasia in group-1 dogs; however, among those reporting, female predominance was significant ($P < 0.025$). In contrast to the group-1 dogs (general diagnostic survey), in group 2 (follow-up subgroup), 4 of the 5 dogs were males, effectively reversing the gender predilection observed previously.

The proportion of hemangiosarcoma to nonangiomatous/nonlymphomatous tumors in our study was 2:1 (group 1), 1.2:1 (group 2), and 1.4:1 (group 3), compared with 4:1 in another study.[16] We found a wide variety of sarcomatous lesions among the dogs in the 3 data groups. The combined prevalence of nonangiomatous/nonlymphomatous sarcomas was similar to that of hemangiosarcoma alone, and these tumors constituted a major cause of splenomegaly, splenectomy, and mortality.

On the basis of our findings, splenic disease is an important clinical consideration in dogs. Pathologic evaluation of specimens submitted because of splenomegaly and other clinical indications for splenectomy are likely to reveal a large nonneoplastic nodular spleen with good prognostic characteristics. The finding of small, red, disseminated intraabdominal masses, when associated with a microscopic diagnosis of splenic hemangiosarcoma, is indicative of a grave prognosis. However, similar lesions of no clinical consequence also can be seen with a diagnosis of splenic hematoma. When survival of the dog is correlated with the type of splenic disease (group 2), a distinctive trend is noticed. These data suggested a markedly different prognosis for canine splenic diseases that are surgically and clinically indistinguishable, emphasizing the importance of careful gross evaluation (by a pathologist, if available) and selection of multiple tissue sections from the interface area between splenic nodules and adjacent splenic parenchyma. The general prognosis for dogs with nodular splenomegaly appears to be better than is generally perceived.

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